

Table 1. DOSAGE

Group		Dosage (µg/ml)
Control	No drug	-
5	Low-dose	25
10	High-dose	40

[0049] After the 24 hours of incubation, an equal volume of 10% formalin phosphate-buffered saline containing 0.2% crystal violet was added to each well and left at room temperature for 20 minutes. The plates were then washed twice with distilled water and dried at room temperature. The absorbency of the stained cells at 590nm was then measured using an automatic microtest-plate reader.

Average absorbency of the control wells (A_c) without any treatment was calculated, average absorbency of each treatment group (A_{Ti}) was determined, and then the average cell viability of each treatment group (V_i) was derived using the following formula:

$$V_i(\%) = \frac{A_{Ti}}{A_c} \times 100\%$$

B. Result

Table 2. CANCER CELL VIABILITY (%)

Group		Absorbency of Stained Cells (M ± SD)	V(%)	tTest w/ Control	tTest w/ Rh2
5	Control	No drugs	.368 ± .069	100.00	
	Low-dose (25µg/ml)	Rh2	.278 ± .030	78.49	P<0.01
		PAM-120	.220 ± .051	62.08	P<0.01
		PBM-100	.223 ± .040	62.72	P<0.01
		PAN-30	.249 ± .045	70.30	P<0.01
10	High-dose (40µg/ml)	Rh2	.181 ± .049	50.99	P<0.01
		PAM-120	.125 ± .031	35.34	P<0.01
		PBM-100	.130 ± .019	36.51	P<0.01
		PAN-30	.147 ± .032	41.49	P<0.01

20 [0050] The results in Table 2 show a significant inhibitory effect on proliferation of H460 cells by each of the novel compounds PAM-120, PBM-100 and PAN-30 (P<0.01 compared with that of the Rh2 control), and a notable increase in inhibitory effect of PAM-120 and PBM-100 on the proliferation of H460 cells (P<0.05 compared with that of Rh2).

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Example 4: Tumor Weight Test

A. Method

30 [0051] Forty (40) C57BL/6J mice weighing 18-22 g were randomly divided into four groups: one control group and three treatment groups, each with 10 animals. Mouse sarcoma 180 cells were hyperdermically transplanted into the mice by using a transplantation needle under the right armpit. After the transplantation, all mice formed a tumor. The mix composition of ginsenosides and sapogenins

including the three novel dammarane sapogenins (PAM-120, PBM-100 and PAN-30), derived as an intermediate product from the process described in Example 2, was prepared into a suspension form. The mice were weighed daily prior to drug administration to determine the actual measurement of drug administered. The drug administration started from 24 hours post tumor transplantation. The mice in the three treatment groups were orally given the mix composition at a daily dose of 0.4 mg/kg, 1.2 mg/kg and 3.6 mg/kg respectively for 8 days using a gastric catheter. The mice in the control group were orally given a normal saline placebo. 24 hours after the last administration of the drug, the mice were sacrificed with an overdose of anesthetics. The weight of the sarcoma in each mouse was measured. The average tumor weight of each treatment group (W_{t_i}) and that of the control group (W_c) were calculated, and the tumor inhibition ratio (R_i) of each treatment group was determined with the following formula:

$$R_i(\%) = \frac{W_c - W_{t_i}}{W_c} \times 100\%$$

B. Result

Table 3. TUMOR WEIGHT RATIO (%)

GROUP	MICE#	Tumor Weight (g) (M±SD)	R(%)	P
Control	10	2.995 ± 0.621		
Mix 0.4 mg/kg	10	1.269 ± 0.525	57.63	<0.01
Mix 1.2 mg/kg	10	0.725 ± 0.270	75.79	<0.01
Mix 3.6 mg/kg	10	0.388 ± 0.130	87.04	<0.01

The results in Table 3 have demonstrated that oral administration of the subject mix composition, the intermediate product from Example 2, achieves tumor inhibition ratios of 58%, 76% and 87% respectively at the doses of 0.4 mg/kg, 1.2 mg/kg and 3.6 mg/kg, showing a dose related anti-cancer efficacy of the mix of the intermediate product of Example 2 containing the three novel sapogenins PAM-120, PBM-100 and PAN-30 and some other saponins and sapogenins whose structures are known or unknown.